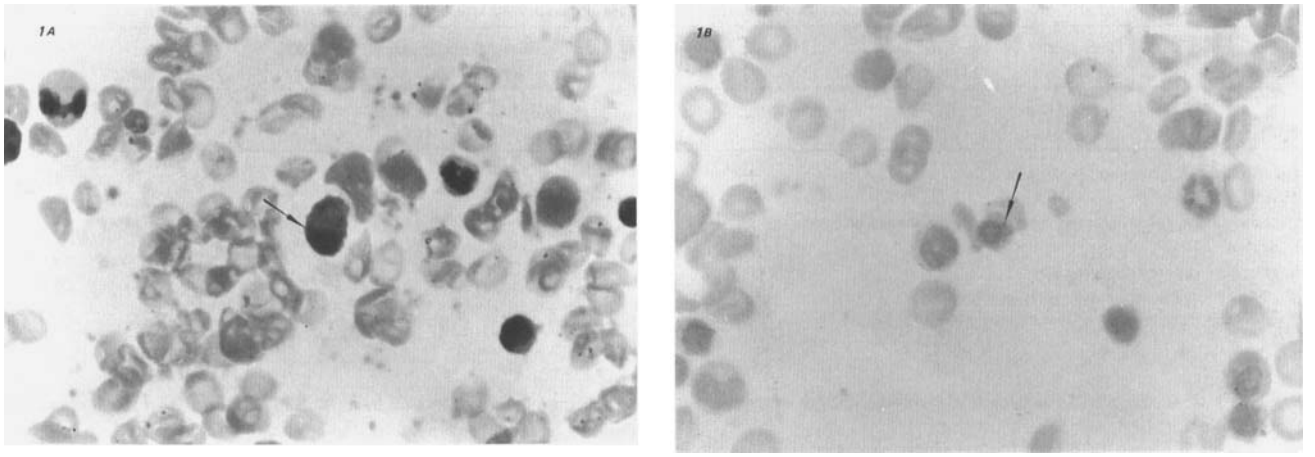

Fig. 1. A: Bone marrow smear stained by the May-Grünwald-Giemsa method. Arrow, iron granule. (High-magnification, ×1,000.) B: Bone marrow smear stained by Perls' Prussian blue and counter-stained with safranin 0.1%. This stain verifies the hemosiderin nature of the granules. Arrow, iron granule. (High magnification, ×1,000.)

Grünwald-Giemsa bone marrow smears (Fig. 1A), but they are blue in Perls' Prussian blue stain with safranin 0.1% counterstain (Fig. 1B).

In 1991 we reported our experience with two male patients, both with excessive drinking habits and macrocytic anemia without megaloblasts; one patient also had liver cirrhosis [2]. Now we report on the presence of iron granules in plasma cells, quantitated as grade 1 [3], in a 91-year-old female patient admitted for anorexia and asthenia. The patient had a previous significant history of breast cancer removed surgically (quadrantectomy). Laboratory tests showed a macrocytic anemia, with mild leucocytopenia. Bone marrow aspiration did not show megaloblastosis. Nodular biopsy on the breast scar showed the presence of neoplastic cells.

We refer to the morphologic aspect because only a few cases have been reported, although the technique employed for identification is easy to use. Both the source of the phenomenon and the causal mechanism are unknown.

It has not yet been determined whether the presence of iron granules in plasma cells could be the expression of a specific nosologic entity.

Guido D'Angelo
Paolo Cueroni
Laboratorio di Chimica-Clinica, Ematologia e Microbiologia,

Ivano Cosini
Unità di Medicina, Ospedale A.Bellini -21019, Somma Lombardo, Varese, Italy

References

1. Jabbe JB: The reticulo-endothelial system. In Downey NY (ed): "Handbook of Hematology." New York: 1938, p. 1170.
2. D'Angelo G, Giardini C, Zanco MD: With regards to the presence of iron granules in plasma cells. Rec Prog Med 82:675–676, 1991.
3. McCurley TL, Cousar JB, Graber SE, Glick AD, Collins RD: Plasma cells iron and morphologic features. Am J Clin Pathol 81:312–316, 1984.

Homozygous Thalassemia and Difficult Endotracheal Intubation

To the Editor: Difficulty in airway management constitutes and essential predisposing factor of morbidity and mortality attributable to anesthesia, especially when it is not anticipated preoperatively [1]. With this in mind, we investigated the epidemiological characteristics in difficult endotracheal intubation in regard to the presence of protruding maxilla in homozygous thalassemia patients.

Data were collected in a series of 5,166 anesthetic case records of consecutive adult patients undergoing general anesthesia for routine surgery. Fifteen specialist anesthetists carried out the routine preoperative airway assessment using standardised guidelines. Table I shows eight individual risk factors implicated to cause difficulty in intubation [1–3]. Hypertrophy of the maxilla was defined as forward protrusion of the upper incisors beyond the lower incisors. Anesthesia was induced intravenously; 1 min after administration of succinylcholine 1.5 $mg \cdot kg^{-1}$ tracheal intubation was carried out using a Macintosh laryngoscope, blade #3 or 4. Severity of difficulty in intubation was estimated according to the view obtained at laryngoscopy [4] (arytenoids and/or glottis = easy; only epiglottis or not even epiglottis = difficult).

Homozygous thalassemia patients had a notable prevalence in the series studied (58/5,166; 1.1%); however, it was not indicative of the general population [5], as our hospital is a referral center for the disease. It is widely accepted that hemoglobin levels are inversely correlated with maxilla size. According to our findings, the relative prevalence of patients with no evidence of hypertrophy of the maxilla was 26/58 (44.8%), reflecting the effectively followed-up homozygous thalassemia patients. Statistical analysis revealed a highly significantly increased risk of difficult intubation amongst patients presented with hypertrophy of maxilla due to thalassemia, as compared to patients with no evidence of any risk factor (Table I, probability of difficulty: 18.8% vs. 0.9%, two-tailed P-value = 0.0017, Fisher's exact test; relative risk: 20.7, $9.5 < RR < 45.2$, 95% Taylor series confidence limits).

In conclusion, homozygous thalassemia, when accompanied by maxillary deformity, constitutes an aggravating factor for difficult intubation. It proved to be of statistically equal strength when compared to traditionally recognised risk factors (Table I).

Gregory S. Voyagis
Kyriakos P. Kyriakis
Department of Anesthesiology, Laikon General Hospital, Athens, Greece

References

1. Benumof JL: Management of the difficult airway. Anesthesiology 75:1087, 1991.
2. Otto CW: Tracheal intubation. In Nunn JF, Utting JE, Brown BR (eds): "General Anesthesia" Ed 5. London: Butterworths, 1989.

TABLE I. Probability of Difficult Endotracheal Intubation by Results of Preoperative Airway Assessment[†]

Protruding maxilla (thalassemia)	Prominent upper teeth	Head and neck mobility <90°	TMD[a] <6.5 cm	Long narrow mouth	Airway pathology[b]	Obesity BMI[c] ≥34	IG[d] <3.5 cm	Multiple risk factors	Absence of risk factors
6/32 (18.8)	5/37* (13.5)	11/54* (20.4)	17/60* (28.3)	4/28* (14.3)	19/86* (22.1)	14/156** (9.0)	9/12** (75.0)	25/64 (39.1)	42/4,637 (0.9)

[†]Numbers in parentheses are percentages.
[a]Thyromental distance.
[b]Extremely large goiter and other causes of deviation and/or stenosis of larynx or trachea.
[c]Body mass index.
[d]Interincisors gap (mouth opening).
*χ^2, Nonsignificant, as compared with thalassemia.
**χ^2, $P < 0.001$, as compared with thalassaemia.

3. Wilson ME, Spiegelhalter D, Robertson J: Predicting difficult intubation. Br J Anaesth 61:211, 1988.
4. Cormack RS, Lehane J: Difficult tracheal intubation in obstetrics. Anaesthesia 39:1105, 1984.
5. Fessas P: Prevention of thalassaemia and haemoglobin S syndromes in Greece. Acta Haematol 78:168, 1987.

An Unusual Case of Untreated Chronic Lymphocytic Leukemia Patient Presenting With *Rhodococcus equi* Bacteriemia

To the Editor: The emerging role of rare opportunistic microorganisms in immunocompromised hosts was recently reported in your journal [1]. We present the case of a human immunodeficiency virus (HIV)-negative untreated chronic lymphocytic leukemia (CLL) patient (stage IV Rai classification) with *Rhodococcus equi* (RE) bacteriemia at presentation. RE, a well-described veterinary pathogen, is an intracellular facultative, gram-positive, partially acid-fast coccobacillary microorganism that in immunocompromised human patients may be responsible for bacteremia and invasive pulmonary, extrapulmonary, and disseminated infections [2]. Most cases were recorded among HIV-positive patients and in hemolymphopathic subjects after chemotherapy [3].

A 57-year-old married man was admitted with a 2-month history of abdominal tension and distalis edema because of the occasional finding of hyperleukocitosis (660×10^{-3} μl). He was a retired nurse and had had no apparent exposure to RE sources of infection. On physical examination, he was found to have significant polydistrictual lymphoadenopathies, hepato- and splenomegaly. Pulmonary examination and chest radiography revealed a right pleural effusion. Blood cell differential demonstrated absolute lymphocitosis (L 98%) with diffuse bone marrow substitution. By flow cytometry a typical immunological CLL pattern (CD5+/CD19+, CD23+, HLA-DR+ with dim surface immunoglobulin expression) was demonstrated. CD4+ lymphocytes were 6,460/μl. IgG nephelometric determination was 392 mg/dl (normal range 614–1,290 mg/dl). Because of a single febrile episode (>38°C), the patient underwent a blood microbiological culture with the unexpected isolation of the RE. After counseling, the patient was tested for HIV antibody by enzyme-linked immunosorbent assay (ELISA) and found to be negative. The patient was HBV negative and HCV positive. On the basis of the antibiogram, the patient was treated for 2 months with trimethoprim/sulfamethoxazole. No fever was observed during the entire period, and all subsequent blood and sputum cultures resulted negative. Two months later, following 2 COP (cyclophosphamide, vincristine, and prednisone) and one CNOP (COP plus mitoxantrone) chemotherapeutic regimens, the patient died of severe hepatic failure. No autopsy was done.

Few cases of RE infection have been reported in HIV-negative patients; these cases were previously treated hemathologic patients. To our knowledge, this is the first case of an untreated CLL patient presenting with RE infection. In our opinion, the importance of this case is related to the role of rare opportunistic pathogens in hemolymphopathic disorders both at diagnosis and during treatment. Hematological patients, and in particular CLL patients, are by definition immunodeficient subjects in which pulmonary infections and septicemia represent the principal causes of death. The importance of considering rare opportunistic infections, and in particular RE infection, in hematological patients [4] is also related to a possible frequent occupational and leisure time exposition to RE (contact with horses, sheep, pens, and animal manure in the activities of farmers and gardeners).

Isolated bacteriemia may be a manifestation of latent infection during a period of immunologic depression. RE diagnosis is often delayed because of an insidious course of the infection and of the difficult microbiological identification, since it may be mistaken for a difteroid or occasionally for a mycobacterium based on acid fast resistance. The efficacy of the therapy is strictly related to a rapid diagnosis; it depends on prolonged treatment and on the use of lipophilic antimicrobial drugs that can penetrate the macrophages or neutrophils in which the organisms survive [5]. In conclusion RE infection should be considered an important emerging opportunistic infection in both untreated and treated hematologic patients.

Acknowledgments

This work was supported, in part, by ACRO project (CNR-Rome) and MURST.

Gian Matteo Rigolin
Renato Bigoni
Romedio Spanedda
Gianluigi Castoldi
Institute of Hematology, University of Ferrara, Ferrara, Italy
Maria Rita Rossi
Laboratorio Analisi Chimico-Cliniche e Microbiologia Arcispedale S. Anna, Ferrara, Italy

References

1. Durand JM, Rousseau MC, Gandois JM, Kaplansky G, Mallet MN, Soubeyrand J: Streptococcus lactis septicemia in a patient with chronic lymphocitic leukemia. Am J Hematol 50:64, 1995.
2. Prescott JF: *Rhodococcus equi*: An animal and human pathogen. Clin Microbiol Rev 4:20, 1991.
3. Verville TD, Huicke MM, Greenfield RA, et al: *Rhodococcus equi* infections of humans. 12 cases and a review of the literature. Medicine (Baltimore) 73:119, 1994.
4. Dig C, Gill J, Church DL: *Rhodococcus equi*—an easily missed opportunistic pathogen. Scand J Infect Dis 23:1, 1991.
5. Hondalus MK, Mosser DM: Survival and replication of *Rhodococcus equi* in macrophages. Infect Immun 62:4167, 1994.